Operative findings on microsurgical exploration of the cerebello-pontine angle in trigeminal neuralgia

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SUMMARY   The anatomical findings in 52 patients undergoing posterior fossa exploration for idiopathic trigeminal neuralgia are described. Anatomical abnormalities in the cerebello-pontine angle were found in 46. The commonest was an arterial loop indenting or distorting the nerve at the root entry zone. In view of this the operative procedure has been changed from root section to microvascular decompression.

Although trigeminal neuralgia can occur in patients with multiple sclerosis[1] and with tumours of the cerebello-pontine angle,[2] in the majority the condition has been considered to be idiopathic. In 1934 Dandy[3] first drew attention to the finding of anatomical and pathological structures impinging on the trigeminal nerve in the cerebello-pontine angle in 60% of 215 patients with apparently idiopathic trigeminal neuralgia, but it was 25 years before Gardner[4][5] confirmed the findings and performed the first operation to dissect the compressing artery off the trigeminal nerve.[4] Jannetta[6][7] has since developed this operation to relieve trigeminal neuralgia, but only he and Apfelbaum[8] have reported large series of such microvascular decompressions. Several smaller series have described the operative findings in the cerebello-pontine angle[9-13] but the concept of an anatomical or pathological structure compressing the nerve and causing trigeminal neuralgia remains controversial. This paper describes the operative findings in the cerebello-pontine angle in 52 patients with clinically idiopathic trigeminal neuralgia and discusses these findings in relation to the treatment used.

Patients and methods

The patients were treated between June, 1972 and November, 1982. The sex, side and divisions involved, together with the age at onset and length of history before operation, are given in table 1. Pre-operative vertebral angiography

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was performed in 26 earlier patients and computed tomography (CT) in 24 later patients. Two patients had both investigations. All patients had taken carbamazepine but 41 had ceased to respond to maximum doses and 11 had developed side effects preventing continued treatment. The surgical procedure consisted of exploration of the cerebello-pontine angle through a small retro-mastoid craniectomy with the patient in the lateral decubitis position with hyperventilation anaesthesia. The operating microscope at magnifications of ×10 and ×16 was used for all cases and photographs were taken of the operative findings in 22.

Operative findings

These are detailed in table 2. Arterial loops were found in contact with the trigeminal nerve, usually indenting and displacing the nerve, in 37 (71%). In two other patients arterial loops were found close to the nerve but not in contact. The commonest vessel involved was the superior cerebellar artery (SCA) in 25 patients, usually making a downward loop medial to the nerve near the back of the petrous bone and then turning back upwards, often as a double loop, and producing compression at the point where the nerve enters the side of the pons. The compression at this point was often from the medial side, the distal part of the vessel then turning away upwards and posteriorly. Because the SCA usually lay a little deeper in the operative field than the nerve, it was not always immediately recognised when the nerve was exposed. An upward looping anterior inferior cerebellar artery was identified as the compressing vessel in two cases, while in seven the vessel could not be identified. Ectatic arteries were responsible for compression in three instances, two cases being caused by the basilar artery and one by the contralateral vertebral artery. Four patients were found to have veins indenting or displacing the nerve

Table 1 *Clinical data in 52 patients with trigeminal neuralgia treated by posterior fossa exploration*

Sex		Side		Divisions involved						
M	F	R	L	1	1 + 2	1 + 2 + 3	2	2 + 3	3	
22	30	29	23	2	3	8	14	17	8	

Age of onset (years)							
11–20	21–30	31–40	41–50	51–60	61–70	71+	Mean
1	3	6	7	22	11	2	52·5

Length of history (years)						
1–2	3–5	6–10	11–20	21+	Mean	
14	14	16	7	1	6·4	

and there were three clinically unsuspected tumours (one each of meningioma, trigeminal neurinoma and epidermoid tumour). Thickened arachnoid was found in two cases and there were four negative explorations. These four were all in the first 25 patients and it may be that inexperience led to vascular compression or some other abnormality being missed.

Radiological investigations

The radiological investigations were reviewed by a neuroradiologist (HS) without access to clinical information. Twenty-one angiograms were available for review. The two ectatic basilar arteries and a single ectatic vertebral artery were clearly shown. Two angiograms were performed in patients with tumours, but only the epidermoid tumour was demonstrated. Of the remaining 16 angiograms, nine showed the SCA on the side of the neuralgia looping lower than on the opposite side. In 24 patients who had CT scans only the patient with the epidermoid tumour had an abnormal scan.

We now feel that the vertebral angiography is not indicated in the routine pre-operative assessment for microvascular decompression and perform CT scanning with contrast in all patients to exclude clinically unsuspected cerebello-pontine angle tumours.

Discussion

Since Dandy's[3] original description of anatomical and pathological abnormalities occurring in the cere-

Table 2 *Operative findings in the cerebello-pontine angle in 52 patients with trigeminal neuralgia*

		No.	%
Arterial loop in contact, indenting or displacing nerve	Normal artery	34	71·2
	Ectatic artery	3	
Artery not in contact		2	3·8
Tumour		3	5·8
Vein		4	7·7
Thickened arachnoid		2	3·8
No local finding		4	7·7

bello-pontine angle of patients with apparently idiopathic trigeminal neuralgia, there has been considerable controversy as to the significance of the findings. Working without the operating microscope Dandy[3] found arterial loops in 30·7%, veins in contact with the nerve in 14% and tumours in 5·6%. Our findings show arteries in contact with the nerve in 71%, veins in contact in 7·7% and tumours in 5·8%. Other vessels, both arteries and veins, were often found in close proximity to the trigeminal nerve, or sometimes in contact. However, like Jannetta[7] we have not considered this relevant unless contact was at the point of entry of the nerve into the pons, the root entry zone.

These findings are similar to those of Jannetta,[7] who in a series of 411 operated patients with trigeminal neuralgia found arterial contact with the nerve in 82·2%, venous contact alone in 13·9%, and unsuspected tumours in 3·6%. Apfelbaum[8] in a series of 200 had similar findings of 82·2%, 12·5% and 3·0% for arteries, veins and tumours. Provost and Hardy[9] described one case of vascular compression of the trigeminal root while Constans *et al*[12] described a giant aneurysm of the basilar artery causing trigeminal neuralgia. Wegrzyn[13] has described vascular compression in 68·7% of 80 cases of trigeminal neuralgia. The SCA was the responsible vessel in 24 of the 55 cases with vascular compression. There were seven cases with tumours in the cerebello-pontine angle and five had thickened arachnoid.

It has been argued that these findings are purely coincidental and unrelated to trigeminal neuralgia. Adams, Kaye and Teddy[14] performed posterior fossa exploration in 57 patients with trigeminal neuralgia. They saw a vascular structure close to the trigeminal nerve in a large percentage of cases, but considered it relevant in only 11%. They performed microvascular decompression in those six patients and posterior root section in the others. Pertuiset *et al*[10] found arterial compression in only one case out of 14.

Three anatomical studies have been published examining the relationship of nerves and vessels in the cerebello-pontine angle of unselected cadavers without history of trigeminal neuralgia. Sunderland[15]

dissected 210 brains in subjects of whom no clinical details were known. He found the superior cerebellar artery looping downwards and indenting the nerve in two specimens and perforating the nerve in one. In three specimens the posterior inferior cerebellar artery looped upwards and grooved the undersurface of the nerve and in two specimens ectatic basilar arteries reached and compressed the inner margin of the sensory root. Hardy and Rhoton[16] examined 50 nerves in 25 elderly cadavers. In 26 nerves there was contact between the nerve and superior cerebellar artery and in four there was contact with the anterior inferior cerebellar artery. In only six cases was the point of contact at the root entry zone of the nerve and it was uncommon for the arterial contact to produce distortion of the nerve. Mehta, Fatani and Rao[17] studied 60 nerves in 30 elderly cadavers and found five superior cerebellar artery loops and three anterior inferior cerebellar artery loops in contact with the nerve. Distortion of the nerve due to vascular compression was not seen.

These papers suggest that although arterial contact with the trigeminal nerve in the cerebello-pontine angle may be common in unselected cadavers, it is rare to find vascular contact with the nerve at the root entry zone causing indentation or distortion of the nerve. It is this part of the nerve which has been stated to be the important area for causation of trigeminal neuralgia[18] and we believe that vascular compression, indenting or distorting a nerve at this point, is relevant to the trigeminal neuralgia in the patients we have described. It may be argued that our series is a sub-group within the broad spectrum of the condition. However, comparing our patients with a large series of 637 patients with trigeminal neuralgia of Ruge, Brochner and Davis,[19] they are similar in being mainly in the middle and later years of life, having a preponderance of female sufferers, more right sided involvement and a distribution more commonly in the lower part of the face. All had taken carbamazepine but it became ineffective in 41, the remaining 11 stopping the drug because of unacceptable side effects. This also suggests that vascular loops are not found only in a sub-group of patients who are refractory to carbamazepine.

In view of our belief that these anatomical abnormalities are aetiologically relevant we have changed our operative procedure from fractional section in the earlier patients (22 cases) to decompression in the later group (25 cases). Two patients had decompression combined with small fractional sections. The patients with tumours had them removed, although in the case of the trigeminal neurinoma subtotal removal was combined with fractional section. Microvascular decompression was achieved by dissecting the vessel free and interposing

non-absorbent material between the vessel and the nerve. The material we have used is a small piece of cotton gauze, measuring 1 cm × 0·5 cm, sometimes folded double.

The second patient treated by decompression failed to respond and was re-explored one week later. The decompression was found to be inadequate but in view of the age of the patient and that she had undergone a second operation a fractional section was performed. One other patient suffered some pain in the face eight months after decompression. This pain was not typical trigeminal neuralgia and responded to dental treatment. The rest of the patients who underwent microvascular decompression remain pain-free between five years and six months since operation (mean 30 months). There was no mortality from decompression and no sensory loss was produced. Deafness occurred post-operatively on the side of the operation in one patient, and shows no sign of recovering six months after the procedure.

These findings appear to confirm the concept proposed initially by Dandy,[3] first utilised therapeutically by Gardner[4][5] and later extensively by Jannetta[7] of an extrinsic anatomical cause in the cerebello-pontine angle of most patients with trigeminal neuralgia. There is probably also an additional intrinsic cause in the posterior root and progressive loss of myelin with age[20][21] leading to artificial synapses[22] has been suggested. Age may also be a factor in producing vascular contact as the cerebral arteries lengthen and become more tortuous.[18] This vascular contact at the root entry zone where the central myelin sheath is replaced by the Schwann cell layer[18] may increase demyelination as well as act as an irritant to the nerve. King and Barnett[23] using acute lesions of the trigeminal nerve in anaesthetised cats have shown that an afferent impulse in the damaged trigeminal nerve leads to a volley of delayed efferent impulses in the same nerve. Normally a tactile stimulus on the face sends an impulse to the brainstem, leading to a return volley of impulses for each stimulus.[24] If this volley of impulses reaches an artificial synapse at the trigeminal root further impulses may be sent back to the brainstem, resulting in pain until the neuron is exhausted.[24]

Microvascular decompression appears to be an effective and safe treatment for trigeminal neuralgia and is the procedure of choice for fit patients who wish to avoid the risk of unpleasant sensory symptoms which can follow any sensory loss. Relevant structural anomalies such as we have described are likely to be found in most patients. For patients not concerned about sensory loss, and for those who are old, unfit or fearful of operation, percutaneous thermocoagulation will continue to be a very safe

alternative.

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